Journal of Innovations in Pharmaceutical and Biological Sciences (JIPBS)
ISSN: 2349-2759
Available online at https://jipbs.com/index.php/journal

Review article

Nephrolithiasis: A Comprehensive Review of Pathophysiology, Clinical Presentation, and Management

S. B. Bothra[1], Shahajan Shabbir Baig*[1], Barrawaz Aateka Yahya[2]

[1]*Government College of Pharmacy, Ratnagiri, Maharashtra India.*
[2]*Maulana Azad Educational Trust's, Y. B. Chavan College of Pharmacy, Aurangabad, Maharashtra, India.*

Received on: 02/01/2026, Revised on: 24/03/2026, Accepted on: 26/03/2026, Published on: 30/06/2026.

***Corresponding Author: Shahajan Shabbir Baig**, Government College of Pharmacy, Aurangabad (Chhatrapati Sambhajinagar).
Phone No: 9923350939.
Email id: mirzashahajanbaig@gmail.com

Copyright © 2026**:** Shahajan Shabbir Baig *et al.* This is an open access article distributed under the terms of the Creative Commons Attribution Non Commercial-Share Alike 4.0 International License which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

Keywords: Nephrolithiasis, Urolithiasis, Kidney Stones, Calcium Oxalate, Ureteroscopy, PCNL, Metabolic Syndrome, Stone Prevention.

Vol. 13 (1): 83-95, Jan-June, 2026.

DOI: http://doi.org/10.56511/JIPBS.2026.13110

Abstract

Nephrolithiasis affects approximately 12% of the global population with increasing prevalence linked to obesity, metabolic syndrome, and dietary factors. Calcium oxalate and calcium phosphate stones comprise 70–80% of cases, followed by uric acid (10–15%), struvite (10–15%), and cystine stones (<1%). Stone formation involves complex physicochemical processes including supersaturation, nucleation, crystal growth, and retention, with Randall's plaque serving as a critical nucleation site. Clinical presentation typically includes severe colicky pain, hematuria, and urinary symptoms, though asymptomatic cases occur. Non-contrast helical CT represents the diagnostic gold standard with 98% sensitivity. Management has evolved from open surgery to minimally invasive approaches including extracorporeal shock wave lithotripsy, ureteroscopy, and percutaneous nephrolithotomy. Medical expulsive therapy with alpha-blockers facilitates spontaneous passage of ureteral stones ≤5 mm. Prevention strategies emphasize adequate hydration (≥2 L/day), dietary modifications including normal calcium intake with sodium restriction, and pharmacological interventions such as thiazide diuretics and potassium citrate based on stone composition and metabolic abnormalities. Emerging evidence demonstrates strong associations between metabolic syndrome components—obesity, diabetes, hypertension, and dyslipidemia—and increased nephrolithiasis risk, highlighting the importance of comprehensive metabolic evaluation and lifestyle modifications in preventing recurrence. Understanding these multifactorial aspects enables individualized treatment approaches and reduces the 50% recurrence rate observed within 5–10 years.

1. Introduction

Nephrolithiasis, or kidney stone disease, is among the oldest recognized medical conditions, with lithotomy among the earliest documented surgical procedures [1]. The condition is characterized by the presence of crystalline deposits within the renal pelvis or calyces, though stones may form anywhere along the urinary tract. Urolithiasis is increasingly prevalent globally, affecting approximately 12% of the population with significant geographic and demographic variations [2, 3]. In the United States alone, the prevalence has risen from 3.2% in the 1976–1980 National Health and Nutrition Examination Survey to 5.2% in the 1988–1994 survey, representing a substantial increase across all demographic groups except African Americans and Mexican Americans [4].

The economic and healthcare burden of nephrolithiasis is considerable, with approximately 600,000 cases requiring medical intervention annually in the United States alone [5]. The disease demonstrates a male predominance, with a

lifetime prevalence of 10% in men compared to 5% in women, though this gap has narrowed in recent decades due to changing dietary patterns and lifestyle factors [6]. Peak incidence occurs between the third and fifth decades of life, with decreasing rates after age 60. (7) Of particular concern is the high recurrence rate, with 50% of patients experiencing a second stone within 5–10 years and up to 75% recurrence over 20 years without appropriate preventive management [8, 9].

2. Epidemiology and Risk Factors

2.1 Global and Regional Distribution

The global burden of nephrolithiasis varies significantly across geographic regions, with prevalence rates ranging from 2% to 20% depending on socioeconomic conditions, climate, and dietary habits [10]. In India, the prevalence is estimated at 2.3%, affecting approximately 5–7 million people, with northern states reporting higher incidence compared to southern regions [11]. Environmental factors, particularly hot and dry climates, contribute to increased dehydration and urinary concentration, thereby elevating stone risk. Seasonal variation is well-documented, with peak incidence during summer months corresponding to increased insulation and decreased fluid intake [12]. (Figure 1).

2.2 Demographic Factors

Age-related patterns demonstrate that nephrolithiasis predominantly affects individuals between 20 and 50 years of age, with the mean age of presentation decreasing over recent decades from 46.1 years in 1982 studies to approximately 37.3 years in contemporary cohorts [13, 14]. This downward shift in age distribution raises concerns about lifetime stone burden and cumulative healthcare costs. Gender distribution has evolved from historical ratios of 6–8:1 (male:female) to current ratios of approximately 2:1, reflecting changing dietary habits and lifestyle factors, particularly in Western societies [15, 16].

2.3 Major Risk Factors

Multiple interconnected factors contribute to nephrolithiasis development:

- Dietary Factors: Excessive intake of calcium, oxalates (spinach, tomatoes, nuts), animal protein, sodium, and vitamin D supplementation combined with inadequate fluid intake (<2 liters/day) significantly increases stone risk [17, 18].
- Metabolic Disorders: Primary hyperparathyroidism, cystinuria, gout, renal tubular acidosis, and inherited disorders of oxalate metabolism predispose to specific stone types [19, 20].
- Anatomical Abnormalities: Ureteropelvic junction obstruction, horseshoe kidney, medullary sponge kidney, and calyceal diverticula create conditions favorable for stone formation [21].
- Infections: Urease-producing organisms (Proteus, Pseudomonas, Klebsiella, Staphylococcus) promote struvite stone formation through alkalinization of urine. [22, 23].
- Metabolic Syndrome Components: Obesity (BMI >30), type 2 diabetes mellitus, hypertension, and dyslipidemia demonstrate strong associations with increased nephrolithiasis risk [24, 25].

3. Stone Composition and Classification

Urinary stones exhibit diverse chemical compositions, each with distinct etiologies and therapeutic implications (Figures 02 and 03). Accurate stone analysis is essential for targeted prevention and management strategies [26]. (Table 1).

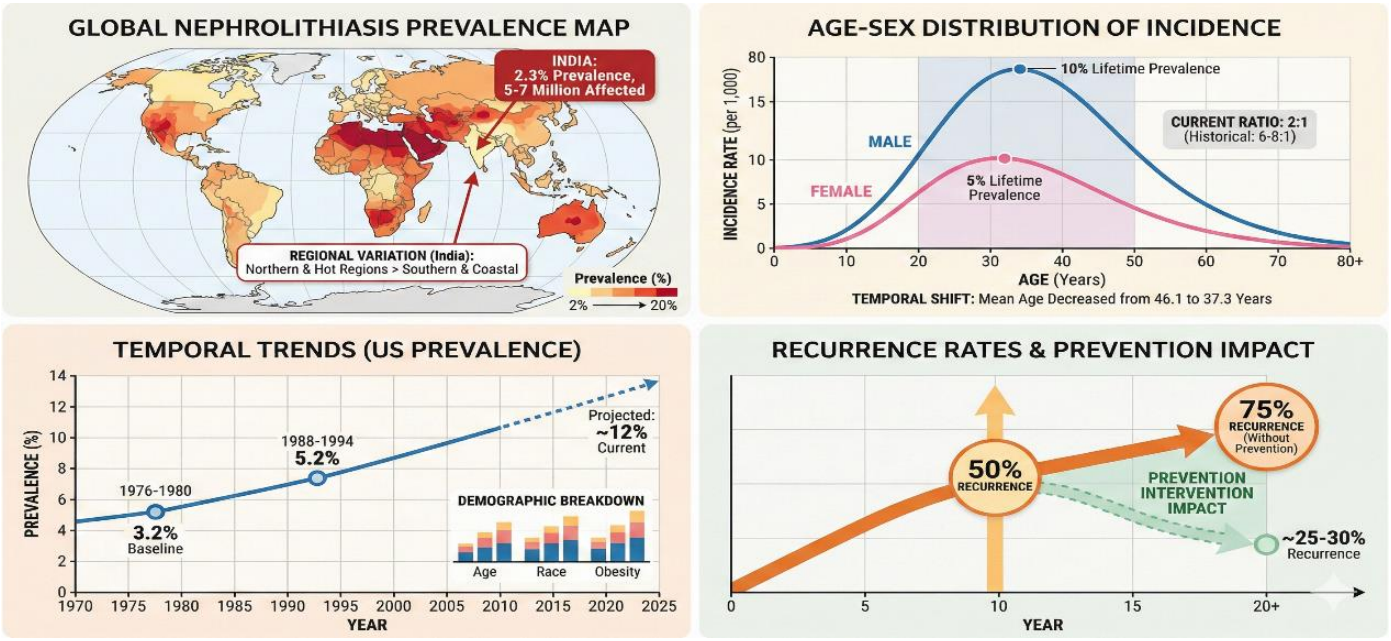


Figure 1. Epidemiological data visualization for nephrolithiasis.

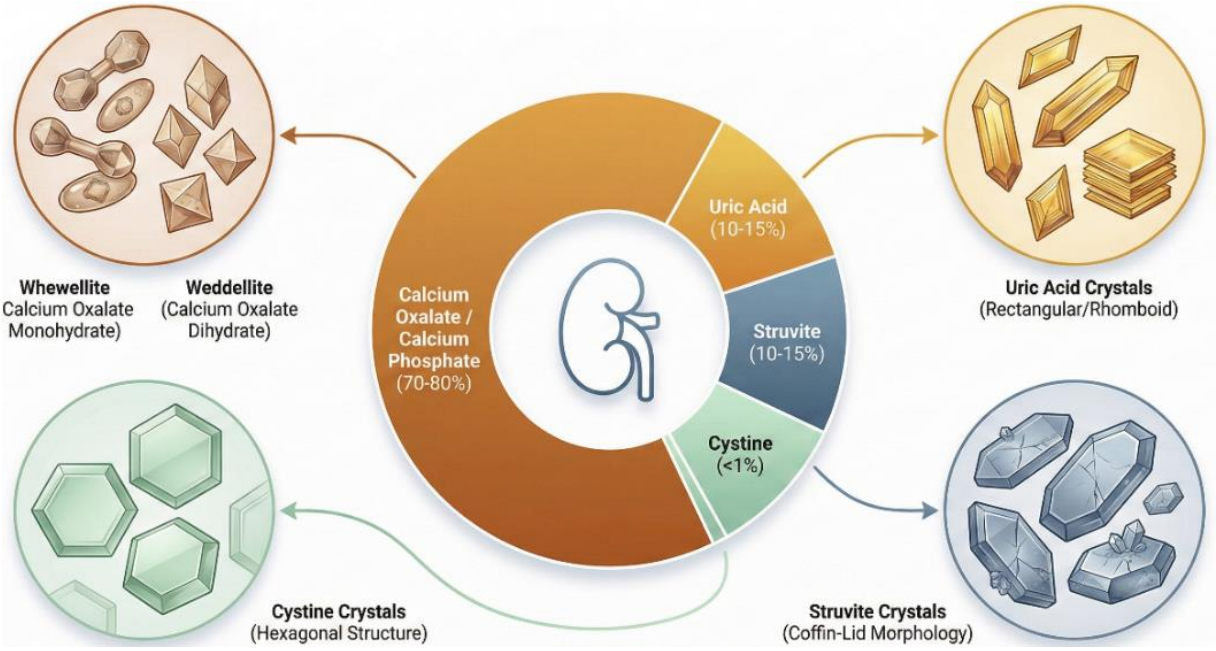


Figure 2. Kidney stone composition and distribution.

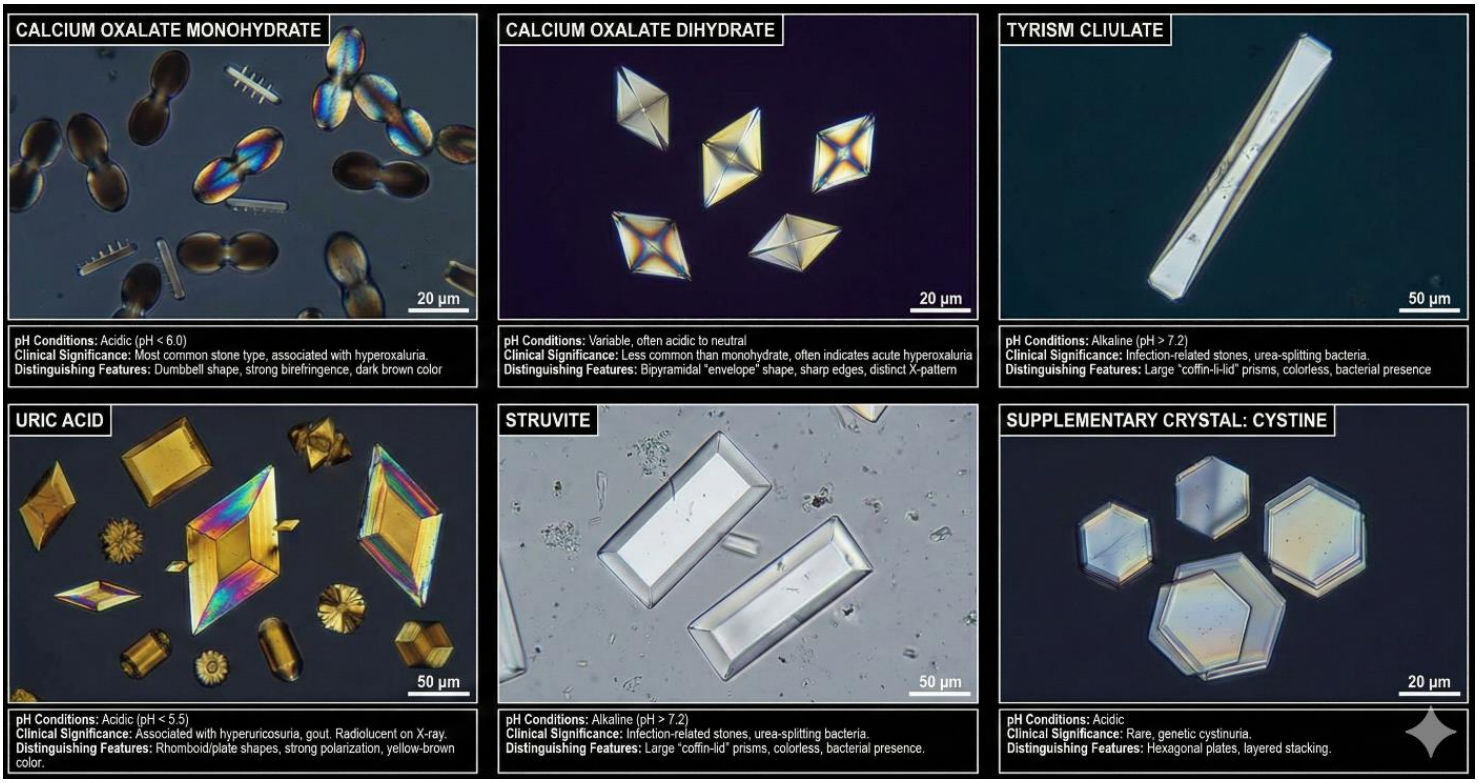


Figure 3. Crystal Morphology.

Table 1. Classification of Urinary Calculi by Composition and Frequency.

Stone Type	Frequency	Causative Factors
Calcium Oxalate/ Phosphate	70–80%	Hypercalciuria, hyperoxaluria, hypocitraturia, hyperuricosuria, metabolic acidosis
Uric Acid	10–15%	Low urinary pH, hyperuricosuria, gout, obesity, metabolic syndrome
Struvite (MAP)	10–15%	Urinary tract infection with urease-producing bacteria
Cystine	<1%	Autosomal recessive cystinuria

3.1 Calcium Stones

Calcium oxalate and calcium phosphate stones represent the predominant stone type, accounting for 70–80% of all nephrolithiasis cases [27]. These stones form through complex mechanisms involving Randall's plaque—subepithelial calcium phosphate deposits on renal papillary surfaces—which serve as nidus for calcium oxalate crystal overgrowth [28]. Hypercalciuria (urinary calcium excretion >300 mg/day in men, >250 mg/day in women) constitutes the primary risk factor, occurring in most calcium stone formers. Idiopathic hypercalciuria may result from increased intestinal calcium absorption, enhanced bone resorption, or renal calcium leak [29, 30]. Paradoxically, dietary calcium restriction is contraindicated as it increases intestinal oxalate absorption and may promote stone formation. Studies demonstrate that normal-calcium diets combined with sodium restriction prove more effective than low-calcium diets in preventing recurrent calcium stones [31].

3.2 Uric Acid Stones

Uric acid stones, comprising 10–15% of urinary calculi, form predominantly in abnormally acidic urine (pH <5.5) rather than from hyperuricosuria alone [32]. The association between metabolic syndrome, insulin resistance, and uric acid nephrolithiasis has gained increasing recognition. Insulin resistance impairs renal ammoniagenesis, resulting in insufficient buffering of secreted hydrogen ions and consequent acidic urinary Ph [33, 34]. Obesity, type 2 diabetes, and metabolic syndrome therefore significantly increase uric acid stone risk. These radiolucent stones require helical CT or ultrasonography for detection, as they are not visible on plain radiography [35]. Treatment focuses on urinary alkalinization to pH >6.0 using potassium citrate or sodium bicarbonate, which may actually dissolve existing stones [36].

3.3 Struvite Stones

Struvite stones, composed of magnesium ammonium phosphate, develop exclusively in the presence of urinary tract infections with urease-producing bacteria, including Proteus, Klebsiella, Pseudomonas, and Staphylococcus species [37, 38] Urease hydrolyzes urea to ammonia and carbon dioxide, alkalinizing urine and creating favorable conditions for magnesium ammonium phosphate crystallization. These stones frequently form staghorn calculi—branched concretions filling the renal pelvis and calyces—and occur more commonly in women and patients with chronic urinary obstruction or neurogenic bladder [39] Management requires complete stone removal combined with antimicrobial therapy, as residual stone fragments harbor bacteria and promote recurrence [40].

4. Pathophysiology of Stone Formation

Nephrolithiasis results from a complex interplay of physicochemical and biological processes. Stone formation requires supersaturation of urine with lithogenic substances, followed by nucleation, crystal growth, aggregation, and ultimately retention within the urinary tract (Figure 04). [41, 42].

4.1 Supersaturation and Nucleation

Supersaturation occurs when the concentration of stone-forming salts exceeds their solubility in urine, creating a thermodynamically unstable solution [43]. Key determinants include urinary volume, pH, and concentrations of calcium, oxalate, phosphate, uric acid, and cystine. Low urine volume represents the single most important modifiable risk factor, as it increases solute concentration across all stone types. Nucleation—the initial formation of microscopic crystals—may occur homogeneously in solution or heterogeneously on existing surfaces such as cellular debris, epithelial cells, or Randall's plaque [44].

4.2 Crystal Growth and Aggregation

Following nucleation, crystals grow through continued precipitation of solutes from supersaturated urine. The rate of crystal growth depends on the degree of supersaturation and the presence of promoters or inhibitors [45]. Crystal aggregation—the clustering of individual crystals—significantly accelerates stone formation, as aggregated particles exceed the size threshold for spontaneous urinary passage. Urinary macromolecules including Tamm-Horsfall protein, nephrocalcin, and uropontin modulate crystal aggregation, with alterations in these proteins contributing to stone susceptibility [46].

4.3 Crystal Retention and Randall's Plaque

For clinically significant stones to develop, crystals must be retained within the urinary tract rather than passing with urine flow. Randall's plaque—interstitial calcium phosphate deposits in the renal papillae—provides attachment sites for calcium oxalate crystals [47, 48]. These plaques form in the basement membrane of thin loops of Henle and extend to the papillary surface, where they serve as heterogeneous nucleation sites (Figure 05). Papillary coverage by Randall's plaque correlates directly with urinary calcium concentration and inversely with urine volume and pH [49]. Additionally, crystal-epithelial cell interactions may promote retention through cellular adhesion mechanisms, particularly in the presence of cellular injury or oxidative stress [50].

5. Clinical Presentation and Diagnosis

5.1 Signs and Symptoms

Nephrolithiasis demonstrates protean clinical manifestations depending on stone location, size, and degree of obstruction. Renal colic—severe, intermittent flank pain radiating to the groin, testicle, or labia—represents the hallmark presentation, occurring in approximately 75% of symptomatic patients [51]. Pain results from ureteral obstruction and distention, with characteristic migration patterns as the stone descends: upper ureteral stones cause flank pain; mid-ureteral stones produce pain radiating to the anterior abdomen; and distal stones manifest with urinary frequency, urgency, and dysuria mimicking lower urinary tract infection [52].

Associated symptoms include nausea, vomiting (occurring in 23.8% of cases), fever suggesting concomitant infection (16.9% of cases), and hematuria (microscopic or gross, present in 91% of symptomatic cases) [53]. Importantly, the absence of hematuria does not exclude urolithiasis, as 9% of patients with confirmed stones demonstrate no blood in urine [54]. Asymptomatic presentations may include silent obstruction discovered incidentally on imaging, persistent bacteriuria, or progressive chronic kidney disease.

5.2 Diagnostic Imaging

Non-contrast helical computed tomography (CT) has emerged as the gold standard for nephrolithiasis diagnosis, demonstrating superior sensitivity (98%) and specificity (100%) compared to alternative imaging modalities [55]. Advantages include rapid acquisition, no requirement for contrast administration, detection of radiolucent stones (including uric acid), identification of stones as small as 1–2 mm, and evaluation of alternative diagnoses [56]. In a series of 1,000 consecutive patients, helical CT identified significant alternative or additional diagnoses in 10% of cases [57]. The technique also allows stone density measurement in Hounsfield units, which correlates with stone composition and may predict response to extracorporeal shock wave lithotripsy [58].

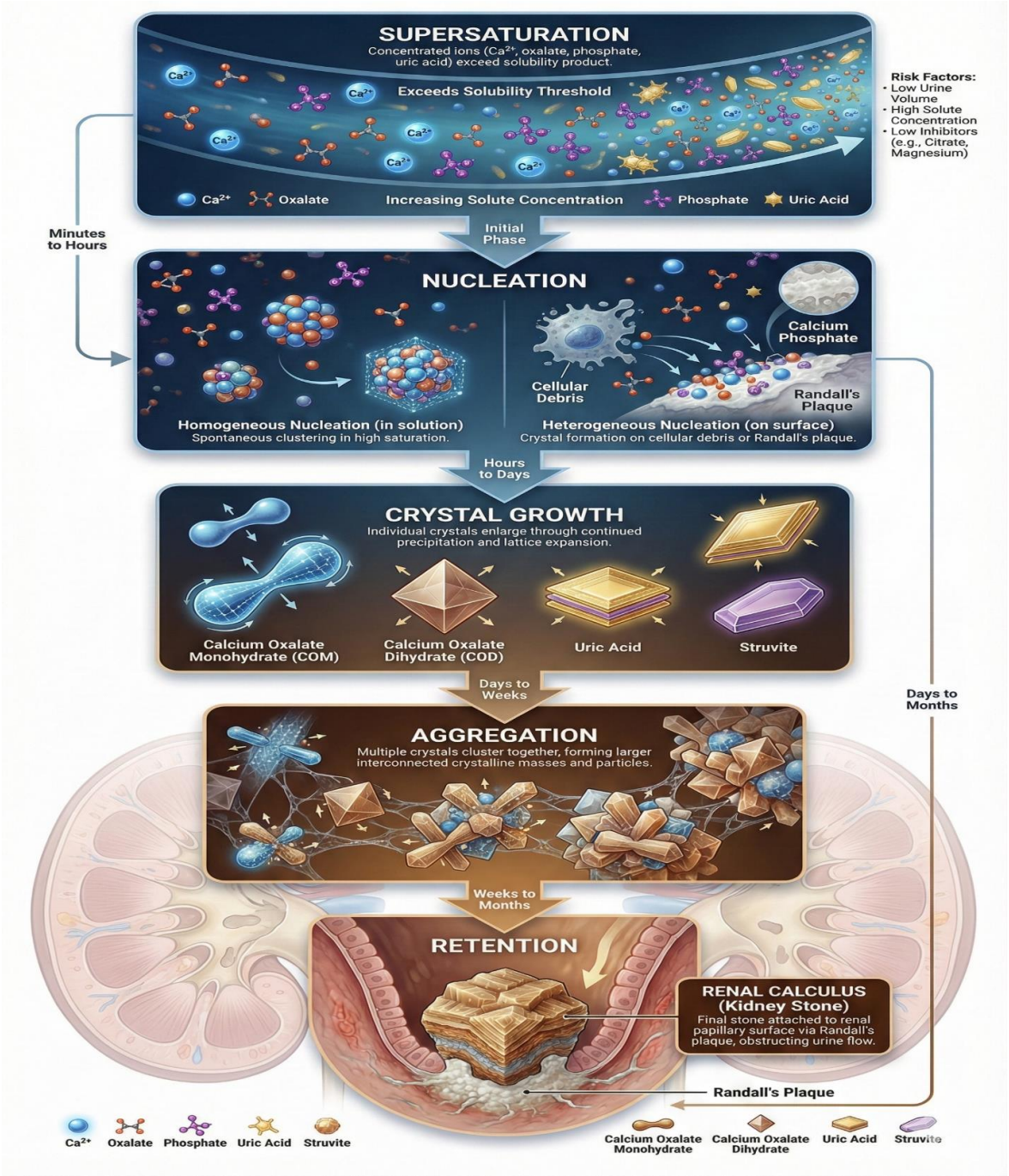


Figure 4. Nephrolithiasis pathophysiology cascade.

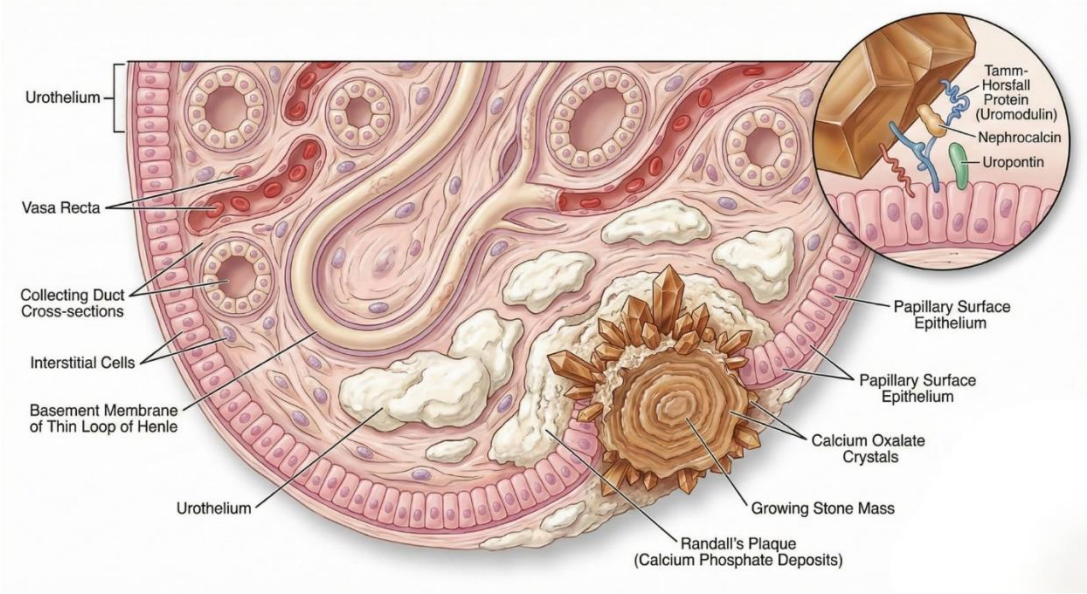


Figure 5. Randall's plaque mechanism in kidney stone formation.

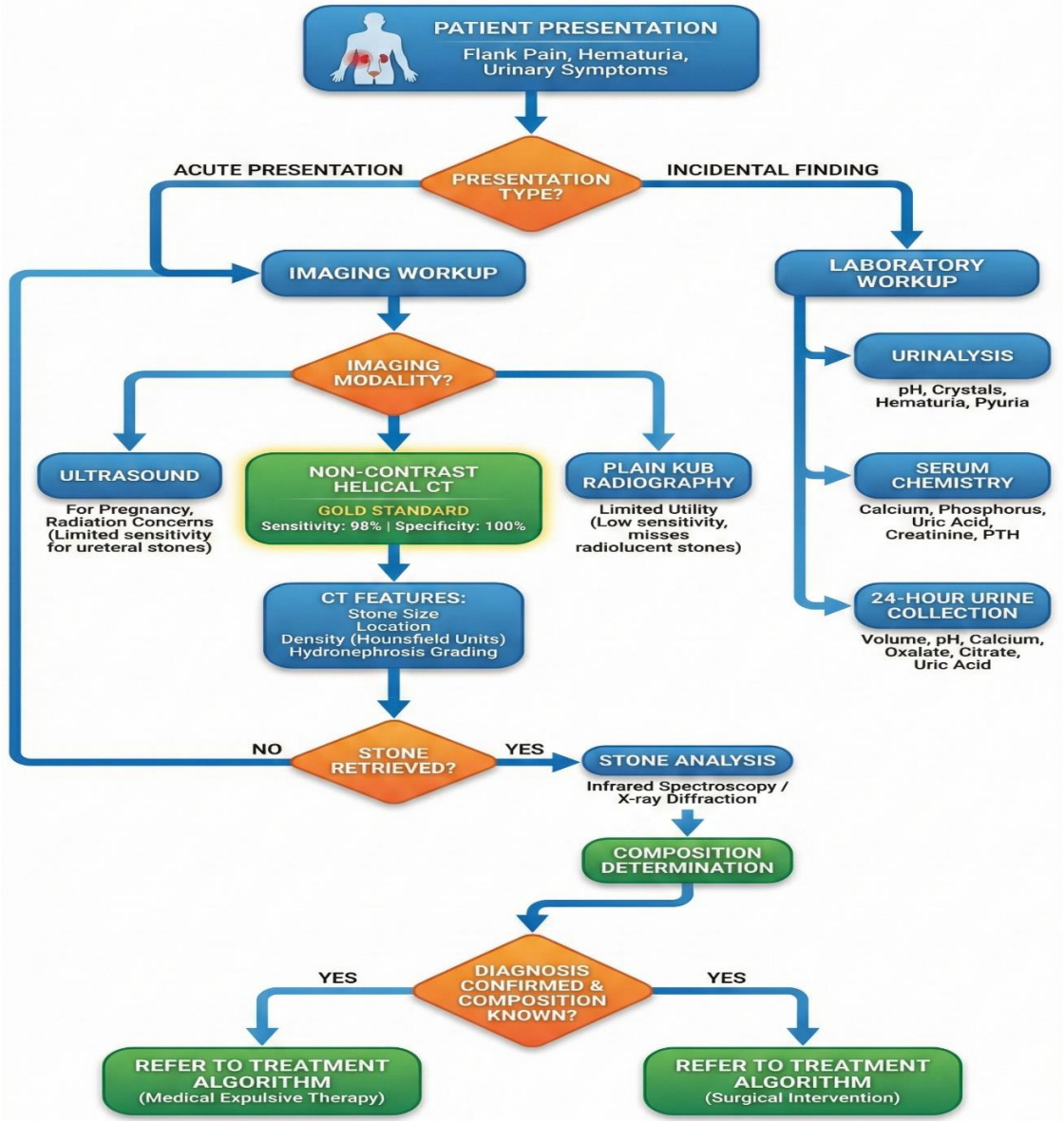


Figure 6. Nephrolithiasis - Diagnostic Algorithm.

Ultrasonography offers advantages including absence of radiation exposure, point-of-care availability, and utility in pregnant patients. However, it demonstrates limited sensitivity (24%) compared to CT and may miss stones smaller than 3 mm [59]. Plain radiography (kidney-ureter-bladder view) provides inadequate diagnostic accuracy due to inability to detect radiolucent stones and overlying bowel gas or bone structures. Intravenous pyelography, historically the standard diagnostic modality, has been supplanted by CT due to inferior diagnostic yield, requirement for contrast administration, and risk of contrast-induced nephropathy. (Figure 06).

5.3 Laboratory Evaluation

Comprehensive metabolic evaluation aids in identifying modifiable risk factors and guiding preventive strategies. Initial assessment includes urinalysis (examining pH, crystals, hematuria, pyuria), urine culture in the presence of infection, and serum chemistry (calcium, phosphorus, uric acid, creatinine, electrolytes). Patients with recurrent stones, young age at presentation (<20 years), strong family history, or specific stone compositions warrant 24-hour urine collection measuring volume, pH, calcium, oxalate, citrate, uric acid, sodium, and creatinine [60, 61]. Elevated parathyroid hormone suggests primary hyperparathyroidism, present in approximately 5% of nephrolithiasis patients [62].

6. Management Strategies

6.1 Acute Management and Medical Expulsive Therapy

Acute stone episodes require prompt pain control and assessment for complications including infection, renal failure, or complete obstruction. Nonsteroidal anti-inflammatory drugs (NSAIDs) provide efficacy equivalent to opioid analgesics for renal colic, with diclofenac demonstrating particular effectiveness in randomized trials [63]. Most stones ≤5 mm pass spontaneously within 4 weeks, with passage rates inversely related to stone size and directly related to distal ureteral location [64].

Medical expulsive therapy facilitates spontaneous stone passage through pharmacological ureteral relaxation. Alpha-1 adrenergic antagonists (tamsulosin 0.4 mg daily) increase stone passage rates, reduce time to passage, and decrease analgesic requirements [65]. A meta-analysis of 693 patients demonstrated that alpha-blockers increase stone passage likelihood with a number needed to treat of 4 [66]. Calcium channel blockers (nifedipine) combined with corticosteroids have shown benefit in randomized trials, though alpha-blockers remain first-line therapy [67]. Adequate hydration (2–3 liters fluid intake daily) promotes urine flow and may facilitate stone descent.

6.2 Interventional Management

The treatment landscape for nephrolithiasis has undergone revolutionary transformation with the development of minimally invasive techniques, which have largely replaced open surgical procedures. Treatment selection depends on stone size, location, composition, and patient factors.

6.2.1 Extracorporeal Shock Wave Lithotripsy (ESWL)

ESWL utilizes focused ultrasonic energy to fragment stones, allowing spontaneous passage of resulting debris. The technique demonstrates optimal efficacy for renal and proximal ureteral stones <20 mm in diameter, achieving stone-free rates of 70–90% for appropriately selected cases [68, 69]. Success correlates inversely with stone size, density, and skin-to-stone distance (obesity). Advantages include non-invasive nature and outpatient administration. Limitations encompass requirement for multiple treatment sessions, incomplete clearance requiring ancillary procedures, and contraindications including pregnancy, bleeding diatheses, and anticoagulation [70]. Complications include perinephric hematoma, acute renal injury, and steinstrasse (accumulation of stone fragments causing ureteral obstruction).

6.2.2 Ureteroscopy (URS)

Ureteroscopy involves retrograde passage of an endoscope through the urethra, bladder, and ureter to directly visualize and treat ureteral or renal stones. Flexible ureteroscopes access all renal calyces, while rigid ureteroscopes provide superior working channel size for lower ureteral stones. Holmium laser lithotripsy fragments stones under direct visualization, with fragments extracted using baskets or allowed to pass spontaneously [71,72]. Contemporary series report stone-free rates of 85–95% for ureteral stones, with success unaffected by stone size up to 10 mm [73]. Advantages include single-session treatment, high success rates, and applicability across all stone types. Routine ureteral stent placement facilitates healing and prevents obstruction from post-procedural edema.

6.2.3 Percutaneous Nephrolithotomy (PCNL)

PCNL represents the gold standard treatment for large renal stones (>20 mm), staghorn calculi, and lower pole stones >10 mm [74, 75]. The procedure involves percutaneous access to the collecting system under fluoroscopic or ultrasound guidance, tract dilation, and nephroscope insertion for direct stone fragmentation and removal. Stone-free rates exceed 95% for appropriately selected cases, surpassing alternative modalities for large stone burden [76, 77]. Complications include bleeding (transfusion rate 5–10%), infection, adjacent organ injury, and prolonged urinary leakage. Hospital stay averages 3–5 days, significantly longer than URS or ESWL. (Figure 07).

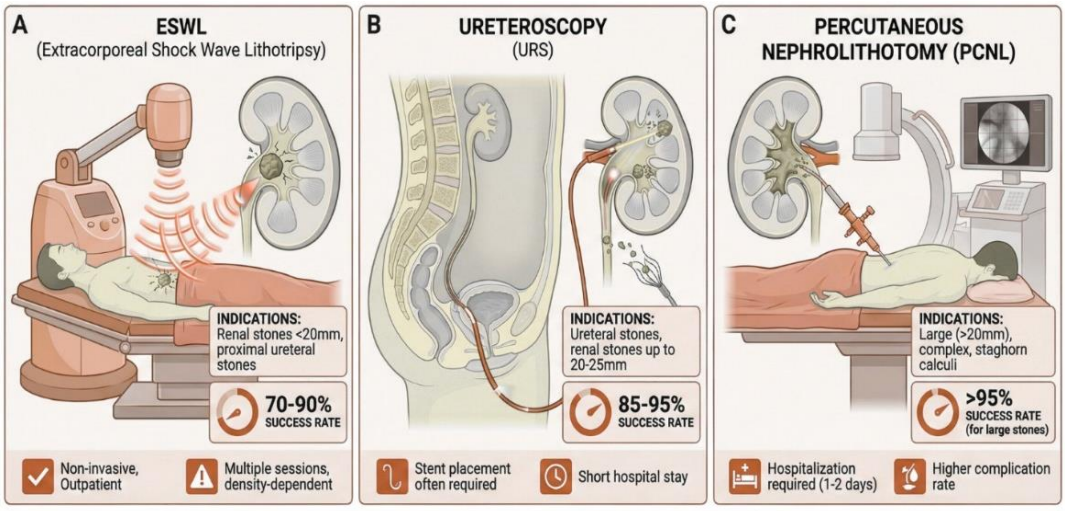
6.3 Prevention of Recurrent Stones

Given the high recurrence rates, prevention strategies constitute a critical component of nephrolithiasis management. Universal recommendations applicable to all stone types include maintaining high fluid intake (sufficient to produce ≥2 liters urine daily), which reduces stone recurrence by approximately 60% [78, 79].

For calcium stone formers, dietary modifications prove essential. Contrary to intuitive reasoning, calcium restriction

is contraindicated and may increase stone risk by promoting intestinal oxalate absorption [80, 81]. Normal calcium intake (1000–1200 mg/day) combined with sodium restriction (<2300 mg/day) and moderate animal protein intake (<2 servings daily) reduces calcium stone recurrence by 50% compared to low-calcium diets [82]. Thiazide diuretics (hydrochlorothiazide 25–50 mg daily or chlorthalidone 25 mg daily) reduce urinary calcium excretion in hypercalciuric patients, decreasing recurrence rates by 40–50% [83]. Potassium citrate supplementation (20–60 mEq daily) increases urinary citrate, alkalinizes urine, and inhibits calcium stone formation [84].

Uric acid stone prevention focuses on urinary alkalinization to pH 6.0–6.5 using potassium citrate or sodium bicarbonate, which may dissolve existing stones. Allopurinol (100–300 mg daily) reduces urinary uric acid excretion in hyperuricosuric patients [85]. For struvite stones, complete surgical removal combined with antibiotic therapy prevents recurrence. Cystine stone management requires aggressive hydration (3–4 liters daily), urinary alkalinization (pH >7.0), and thiol-binding agents (D-penicillamine or tiopronin) in refractory cases [86, 87]. (Figure 08).



Feature	ESWL	URS	PCNL
Stone Size Indications	<20mm, Renal, Proximal Ureteral	<25mm, Ureteral, Renal	>20mm, Complex, Staghorn
Hospitalization Duration	Outpatient	Short Stay (1 Day)	Inpatient (1-2 Days)
Complication Rates	Low (Transient Haematuria, Hematoma)	Moderate (Ureteral Injury, Infection)	Higher (Bleeding, Injury, Sepsis)

Figure 7. Comparative Nephrolithiasis Treatment Modalities.

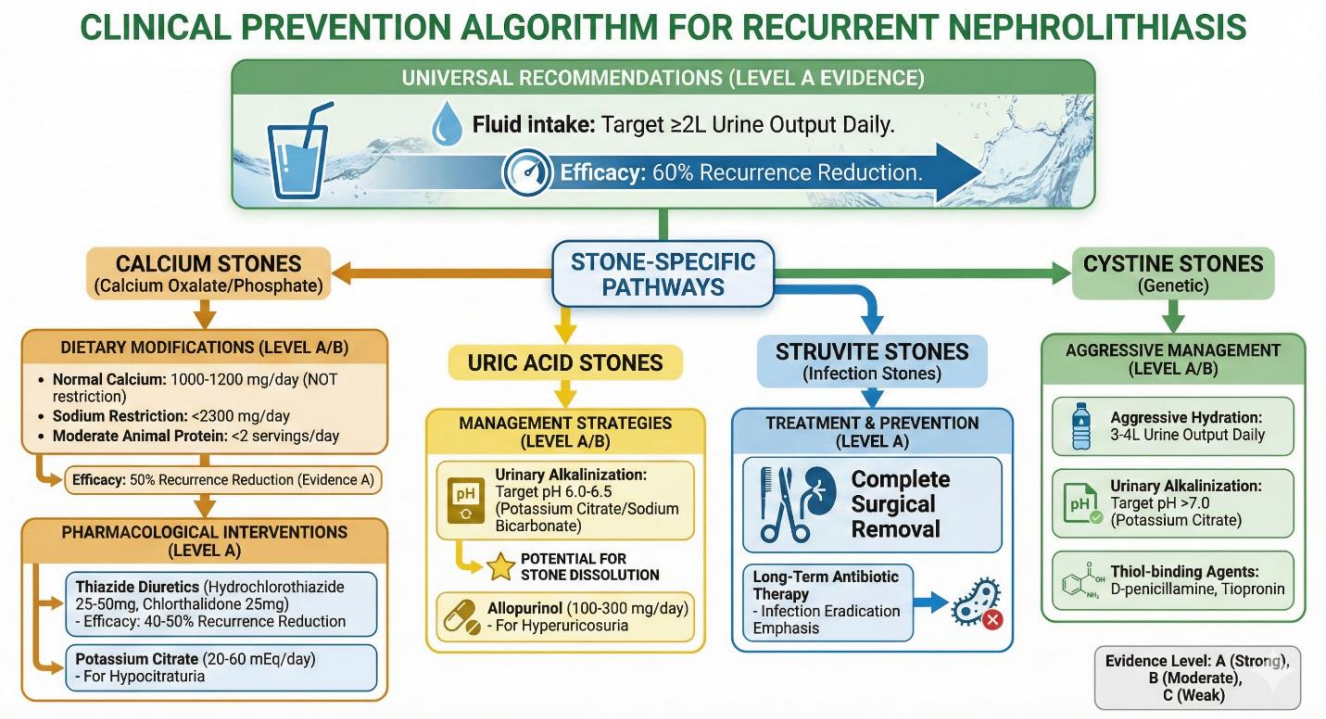


Figure 8. Clinical prevention for recurrent nephrolithiasis.

7. Metabolic Syndrome and Nephrolithiasis

Emerging evidence establishes strong associations between metabolic syndrome and nephrolithiasis, with important implications for both epidemiology and pathophysiology [88, 89]. Metabolic syndrome—defined by the International Diabetes Federation as central obesity (waist circumference >90 cm in men, >80 cm in women) plus two or more of the following: elevated triglycerides (>150 mg/dL), reduced HDL cholesterol (<40 mg/dL in men, <50 mg/dL in women), hypertension (>130/85 mmHg), and impaired fasting glucose (>100 mg/dL)—affects approximately 25–40% of nephrolithiasis patients [90, 91].

Insulin resistance, the hallmark of metabolic syndrome, impairs renal ammoniagenesis, resulting in decreased urinary pH and increased uric acid stone risk [92, 93]. Additionally, metabolic syndrome associates with increased urinary calcium and oxalate excretion, decreased citrate levels, and higher urinary uric acid, creating a lithogenic milieu [94, 95]. Epidemiological studies demonstrate that stone prevalence increases progressively with the number of metabolic syndrome components, from 3% in individuals with zero features to 9.8% with all five components [96]. Each individual component—obesity, diabetes, hypertension, and dyslipidemia—independently increases stone risk [97, 98].

Recent investigations confirm significant correlations between metabolic syndrome and calcium urolithiasis, with stone formers demonstrating higher prevalence of central obesity (60.7% vs 45.0%), elevated triglycerides (34.3% vs 21.4%), and reduced HDL cholesterol (43.6% vs 22.1%) compared to non-stone formers. (99) These findings underscore the importance of metabolic syndrome screening in nephrolithiasis patients and suggest that lifestyle modifications targeting weight reduction, glycemic control, and lipid management may reduce stone recurrence (Figure 09). Furthermore, the bidirectional relationship between kidney stones and metabolic diseases highlights nephrolithiasis as a potential marker for cardiovascular risk, warranting comprehensive metabolic evaluation in affected patients [100].

8. Conclusions and Future Directions

Nephrolithiasis represents a complex, multifactorial disease with increasing global prevalence and significant healthcare burden. Understanding the intricate pathophysiology of stone formation—encompassing supersaturation, nucleation, crystal growth, aggregation, and retention—provides the foundation for rational therapeutic approaches. The evolution from open surgical procedures to minimally invasive techniques has dramatically improved patient outcomes, with ESWL, ureteroscopy, and PCNL offering high success rates with acceptable morbidity.

However, the persistently high recurrence rates emphasize the critical importance of preventive strategies. Comprehensive metabolic evaluation, dietary counseling, pharmacological interventions, and treatment of underlying disorders constitute essential components of long-term management.

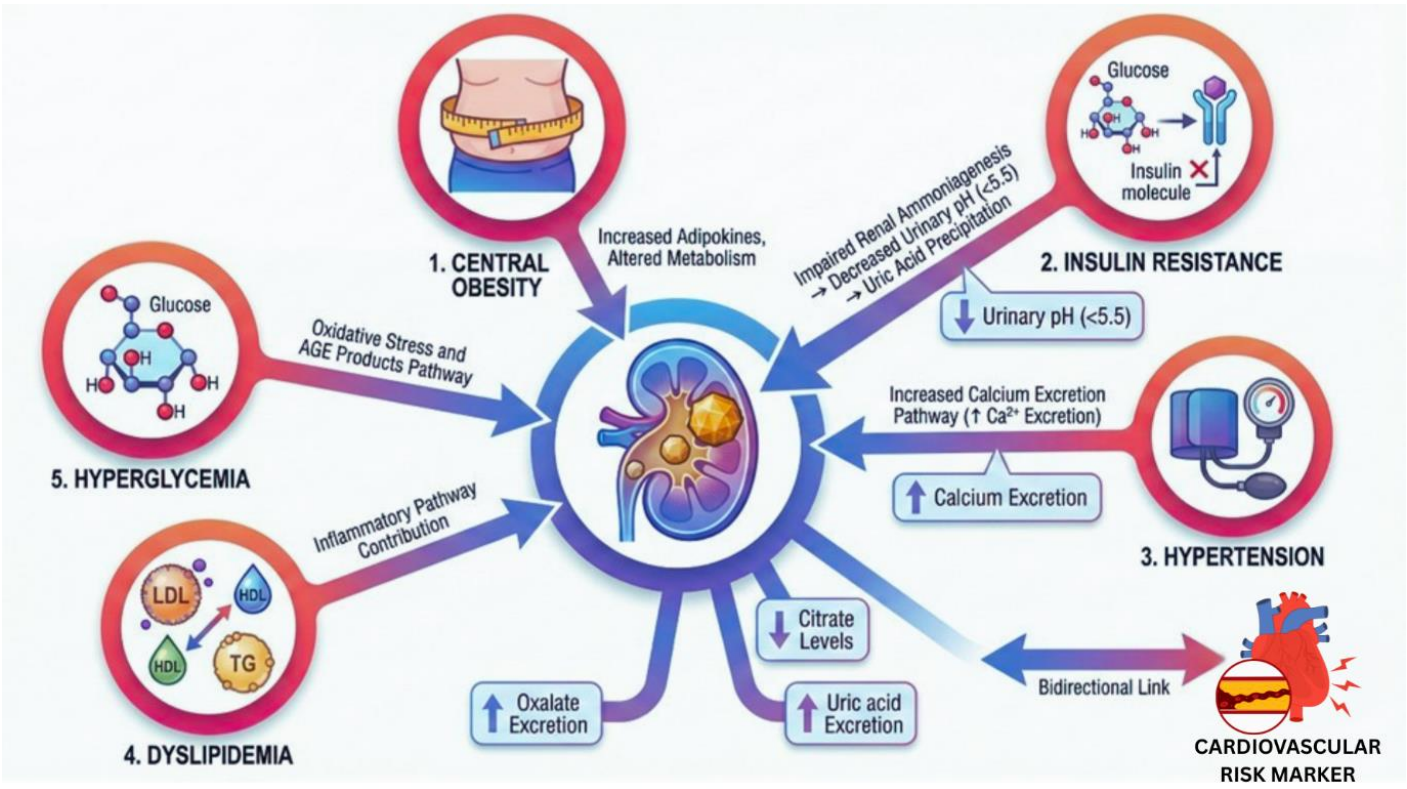


Figure 9. Links between metabolic syndrome and nephrolithiasis.

The emerging recognition of associations between metabolic syndrome and nephrolithiasis opens new avenues for prevention and suggests that lifestyle modifications targeting obesity, diabetes, and dyslipidemia may reduce stone recurrence.

Future research priorities include elucidating molecular mechanisms of stone formation, developing novel pharmacological agents targeting specific pathways, refining risk stratification tools to identify high-risk patients, and implementing cost-effective screening and prevention programs. Additionally, investigation of genetic susceptibility factors may enable personalized approaches to prevention and treatment. As the global epidemic of obesity and metabolic syndrome continues, addressing the growing burden of nephrolithiasis will require integrated, multidisciplinary strategies encompassing primary prevention, early diagnosis, effective treatment, and long-term metabolic management.

Conflict of Interest

The author declares no competing financial interests or personal relationships that could have influenced the work reported in this paper.

Acknowledgements

The authors sincerely thanks the Principal, and staff of Government College of Pharmacy, Aurangabad (Chhatrapati Sambhajinagar), for providing the academic support and resources necessary to carry out this work.

Ethics Approval and Consent to Participate

Not applicable.

Author Contribution

All authors are contributed equally in this work.

Declaration of Generative AI

This manuscript has utilized OpenAI's Cloude 4.6 to enhance the clarity and coherence of the language. The tool was employed exclusively for language improvement purposes with ethical and academic standards. The authors take full responsibility for the content and integrity of the manuscript.

Funding

None.

References

1. Hall, P. M. Nephrolithiasis: Treatment, Causes, and Prevention. Cleve. Clin. J. Med. 2009, 76 (10), 583–591.
2. Romero, V.; Akpinar, H.; Assimos, D. G. Kidney Stones: A Global Picture of Prevalence, Incidence, and Associated Risk Factors. Rev. Urol. 2010, 12 (2–3), e86–e96.
3. Johnson, C. M.; Wilson, D. M.; O'Fallon, W. M.; Malek, R. S.; Kurland, L. T. Renal Stone Epidemiology: A 25-Year Study in Rochester, Minnesota. Kidney Int. 1979, 16, 624–631.
4. Stamatelou, K. K.; Francis, M. E.; Jones, C. A.; Nyberg, L. M.; Curhan, G. C. Time Trends in Reported Prevalence of Kidney Stones in the United States: 1976–1994. Kidney Int. 2003, 63, 1817–1823.
5. Antonelli, J. A.; Maalouf, N. M.; Pearle, M. S.; Lotan, Y. Use of the National Health and Nutrition Examination Survey to Calculate the Impact of Obesity and Diabetes on Cost and Prevalence of Urolithiasis in 2030. Eur. Urol. 2014, 66 (4), 724–729.
6. Hiatt, R. A.; Dales, L. G.; Friedman, G. D.; Hunkeler, E. M. Frequency of Urolithiasis in a Prepaid Medical Care Program. Am. J. Epidemiol. 1982, 115, 255–265.
7. Lancina Martin, J. A.; Rodriguez-Rivera, G. J.; Novas Castro, S.; Rodriguez Gomez, I.; Fernandez Rosado, E.; Alvarez Castelo, L. Metabolic Risk Factors in Calcium Urolithiasis According to Gender and Age of the Patients. Actas Urol. Esp. 2002, 26, 111–120.
8. Morse, R. M.; Resnick, M. I. Ureteral Calculi: Natural History and Treatment in the Era of Advanced Technology. J. Urol. 1991, 145, 263–265.
9. Glowacki, L. S.; Beecroft, M. L.; Cook, R. J.; Pahl, D.; Churchill, D. N. The Natural History of Asymptomatic Urolithiasis. J. Urol. 1992, 147, 319–321.
10. Trinchieri, A.; Coppi, F.; Montanari, E.; Del Nero, A.; Zanetti, G.; Pisani, E. Increase in the Prevalence of Symptomatic Upper Urinary Tract Stones During the Last Ten Years. Eur. Urol. 2000, 37, 23–25.
11. Colobawalla, B. N. Incidence of Urolithiasis in India. ICMR Tech. Rep. Ser. 1971, 8, 42–51.
12. Buchholz, N. P. N.; Abbas, F.; Afzal, M.; Khan, R.; Rizvi, I.; Talati, J. The Prevalence of Silent Kidney Stones—An Ultrasonographic Screening Study. J. Pak. Med. Assoc. 2003, 53 (1), 24–25.
13. Fetter, T. L.; Zimskind, P. D. Statistical Analysis of Patients With Urinary Calculi. JAMA 1961, 186, 21.
14. Dinakar, N.; Shaik, A.; Reddy, N. R.; Swathi, M. A Clinical Study on the Presentation and Management of Urolithiasis. Int. Surg. J. 2024, 11 (3), 1215–1222.
15. Lieske, J. C.; Peña de la Vega, L. S.; Slezak, J. M.; Bergstralh, E. J.; Leibson, C. L.; Ho, K. L.; Gettman, M. T. Renal Stone Epidemiology in Rochester, Minnesota: An Update. Kidney Int. 2006, 69, 760–764.
16. Knoll, T.; Schubert, A. B.; Fahlenkamp, D.; Leusmann, D. B.; Wendt-Nordahl, G.; Schubert, G. Urolithiasis Through the Ages: Data on More Than 200,000 Urinary Stone Analyses. J. Urol. 2011, 185 (4), 1304–1311.
17. Taylor, E. N.; Curhan, G. C. Diet and Fluid Prescription in Stone Disease. Kidney Int. 2006, 70, 835–839.
18. Borghi, L.; Meschi, T.; Amato, F.; Briganti, A.; Novarini, A.; Giannini, A. Urinary Volume, Water and Recurrences in Idiopathic Calcium Nephrolithiasis: A 5-Year Randomized Prospective Study. J. Urol. 1996, 155, 839–843.
19. Parks, J. H.; Coe, F. L.; Evan, A. P.; Worcester, E. M. Clinical and Laboratory Characteristics of Calcium Stone-Formers With and Without Primary Hyperparathyroidism. Br. J. Urol. 2008, 103, 670–678.
20. Danpure, C. J.; Rumsby, G. Molecular Aetiology of Primary Hyperoxaluria and Its Implications for Clinical Management. Expert Rev. Mol. Med. 2004, 6, 1–16.
21. Evan, A. P. Physiopathology and Etiology of Stone Formation in the Kidney and the Urinary Tract. Pediatr. Nephrol. 2010, 25, 831–841.
22. Griffith, D. P.; Osborne, C. A. Infection (Urease) Stones. Miner. Electrolyte Metab. 1987, 13, 278–285.
23. Bichler, K. H.; Eipper, E.; Naber, K.; Braun, V.; Zimmermann, R.; Lahme, S. Urinary Infection Stones. Int. J. Antimicrob. Agents 2002, 19, 488–498.

24. Taylor, E. N.; Stampfer, M. J.; Curhan, G. C. Obesity, Weight Gain, and the Risk of Kidney Stones. JAMA 2005, 293, 455–462.
25. Abdulla, A.; Das, S.; Venugopalan, A. V.; Rajeevan, A. T.; Manikandan, M. The Correlation Between Metabolic Syndrome and Calcium Urolithiasis. Int. Surg. J. 2023, 10 (9), 1477–1483.
26. Coe, F. L.; Evan, A.; Worcester, E. Kidney Stone Disease. J. Clin. Invest. 2005, 115 (10), 2598–2608.
27. Coe, F. L.; Parks, J. H.; Bushinsky, D. A.; Langman, C. B.; Favus, M. J. Chlorthalidone Promotes Mineral Retention in Patients With Idiopathic Hypercalciuria. Kidney Int. 1988, 33, 1140–1146.
28. Evan, A. P.; Lingeman, J. E.; Coe, F. L.; Parks, J. H.; Bledsoe, S. B.; Shao, Y.; Sommer, A. J.; Paterson, R. F.; Kuo, R. L.; Grynpas, M. Randall's Plaque of Patients With Nephrolithiasis Begins in Basement Membranes of Thin Loops of Henle. J. Clin. Invest. 2003, 111, 607–616.
29. Broadus, A. E.; Horst, R. L.; Lang, R.; Littledike, E. T.; Rasmussen, H. The Importance of Circulating 1,25-Dihydroxyvitamin D in the Pathogenesis of Hypercalciuria and Renal-Stone Formation in Primary Hyperparathyroidism. N. Engl. J. Med. 1980, 302, 421–426.
30. Breslau, N. A.; Preminger, G. M.; Adams, B. V.; Otey, J.; Pak, C. Y. Use of Ketoconazole to Probe the Pathogenetic Importance of 1,25-Dihydroxyvitamin D in Absorptive Hypercalciuria. J. Clin. Endocrinol. Metab. 1992, 75, 1446–1452.
31. Borghi, L.; Schianchi, T.; Meschi, T.; Guerra, A.; Allegri, F.; Maggiore, U.; Novarini, A. Comparison of Two Diets for the Prevention of Recurrent Stones in Idiopathic Hypercalciuria. N. Engl. J. Med. 2002, 346, 77–84.
32. Pak, C. Y. C.; Sakhaee, K.; Peterson, R. D.; Poindexter, J. R.; Frawley, W. H. Biochemical Profile of Idiopathic Uric Acid Nephrolithiasis. Kidney Int. 2001, 60 (2), 757–761.
33. Kamel, K. S.; Cheema-Dhadli, S.; Halperin, M. L. Studies on the Pathophysiology of the Low Urine pH in Patients With Uric Acid Stones. Kidney Int. 2002, 61, 988–994.
34. Abate, N.; Chandalia, M.; Cabo-Chan, A. V., Jr.; Moe, O. W.; Sakhaee, K. The Metabolic Syndrome and Uric Acid Nephrolithiasis: Novel Features of Renal Manifestation of Insulin Resistance. Kidney Int. 2004, 65, 386–392.
35. Zarse, C. A.; McAteer, J. A.; Tann, M.; Sommer, A. J.; Kim, S. C.; Paterson, R. F.; Hatt, E. K.; Lingeman, J. E.; Evan, A. P.; Williams, J. C., Jr. Helical Computed Tomography Accurately Reports Urinary Stone Composition Using Attenuation Values: In Vitro Verification Using High-Resolution Micro-Computed Tomography Calibrated to Fourier Transform Infrared Microspectroscopy. Urology 2004, 63, 828–833.
36. Sakhaee, K. Pathogenesis and Medical Management of Cystinuria. Semin. Nephrol. 1996, 16, 435–447.
37. Silverman, D. E.; Stamey, T. A. Management of Infection Stones: The Stanford Experience. Medicine (Baltimore) 1983, 62, 44–51.
38. Jennis, F. S.; Lavan, J. N.; Neale, F. C.; Posen, S. Staghorn Calculi of the Kidney: Clinical, Bacteriological and Biochemical Features. Br. J. Urol. 1970, 42, 511–518.
39. Griffith, D. P. Struvite Stones. Kidney Int. 1978, 13, 372–382.
40. Griffith, D. P.; Gibson, J. R.; Clinton, C. W.; Musher, D. M. Acetohydroxamic Acid: Clinical Studies of a Urease Inhibitor in Patients With Staghorn Renal Calculi. J. Urol. 1978, 119, 9–15.
41. Moe, O. W. Kidney Stones: Pathophysiology and Medical Management. Lancet 2006, 367 (9507), 333–344.
42. Courbebaisse, M.; Prot-Bertoye, C.; Bertocchio, J. P.; Lefevre, G.; Daudon, M.; Prie, D. Nephrolithiasis of Adult: From Mechanisms to Preventive Medical Treatment. Presse Med. 2017, 46 (1), 44–52.
43. Robertson, W. G.; Peacock, M.; Marshall, R. W.; Marshall, D. H.; Nordin, B. E. Saturation-Inhibition Index as a Measure of the Risk of Calcium Oxalate Stone Formation in the Urinary Tract. N. Engl. J. Med. 1976, 294, 249–252.
44. Sakhaee, K.; Maalouf, N. M. Metabolic Syndrome and Uric Acid Nephrolithiasis. Semin. Nephrol. 2008, 28 (2), 174–180.
45. Coe, F. L.; Parks, J. H. Pathogenesis and Treatment of Nephrolithiasis. In The Kidney; Brenner, B. M., Rector, F. C., Eds.; W.B. Saunders: Philadelphia, PA, 2000; pp 1841–1867.
46. Lieske, J. C.; Swift, H.; Martin, T.; Patterson, B.; Toback, F. G. Renal Epithelial Cells Rapidly Bind and Internalize Calcium Oxalate Monohydrate Crystals. Proc. Natl. Acad. Sci. U.S.A. 1994, 91 (15), 6987–6991.
47. Evan, A. P.; Lingeman, J. E.; Coe, F. L.; Shao, Y.; Miller, N. L.; Matlaga, B. R.; Phillips, C. L.; Sommer, A. J.; Worcester, E. M. Crystal-Associated Nephropathy in Patients With Brushite Nephrolithiasis. Kidney Int. 2005, 67, 576–591.
48. Kuo, R. L.; Lingeman, J. E.; Evan, A. P.; Paterson, R. F.; Parks, J. H.; Bledsoe, S. B.; Munch, L. C.; Coe, F. L. Urine Calcium and Volume Predict Coverage of Renal Papilla by Randall's Plaque. Kidney Int. 2003, 64, 2150–2154.
49. Evan, A. P.; Coe, F. L.; Lingeman, J. E.; Worcester, E. Insights on the Pathology of Kidney Stone Formation. Urol. Res. 2005, 33, 383–389.
50. Alelign, T.; Petros, B. Kidney Stone Disease: An Update on Current Concepts. Adv. Urol. 2018, 2018, 3068365.
51. Fink, H. A.; Akornor, J. W.; Garimella, P. S.; MacDonald, R.; Cutting, A.; Rutks, I. R.; Monga, M.; Wilt, T. J. Diet, Fluid, or Supplements for Secondary Prevention of Nephrolithiasis: A Systematic Review and Meta-Analysis of Randomized Trials. Eur. Urol. 2009, 56 (1), 72–80.
52. Nasar, M.; Jabeen, A.; Raza, A.; Begum, M. Integrative Approaches to Hasaat Kuliya (Nephrolithiasis) in Unani and Conventional Medicine. Int. J. Clin. Med. Case Rep. 2025, 4 (1), DOI: 10.31579/2834-8664/063.
53. Prasanna, K.; Reddy, D. R. B.; Rao, T. J. M. A Comprehensive Review on Nephrolithiasis. Int. J. Pharm. Res. Appl. 2024, 9 (1), 1286–1294.
54. Li, J.; Kennedy, D.; Levine, M.; Kumar, A.; Mullen, J. Absent Hematuria and Expensive Computerized Tomography: Case Characteristics of Emergency Urolithiasis. J. Urol. 2001, 165, 782–784.
55. Fielding, J. R.; Steele, G.; Fox, L. A.; Heller, H.; Loughlin, K. R. Spiral Computerized Tomography in the Evaluation of Acute Flank Pain: A Replacement for Excretory Urography. J. Urol. 1997, 157, 2071–2073.
56. Katz, D. S.; Scheer, M.; Lumerman, J. H.; Mellinger, B. C.; Stillman, C. A.; Lane, M. J. Alternative or Additional Diagnoses on Unenhanced Helical Computed Tomography for Suspected Renal Colic: Experience With 1,000 Consecutive Examinations. Urology 2000, 56, 53–57.
57. Katz, D. S.; Lane, M. J.; Sommer, F. G. Unenhanced Helical CT of Ureteral Stones: Incidence of Associated Urinary Tract Findings. AJR Am. J. Roentgenol. 1996, 166, 1319–1322.
58. Gupta, N. P.; Ansari, M. S.; Kesarvani, P.; Kapoor, A.; Mukhopadhyay, S. Role of Computed Tomography With No Contrast Medium Enhancement in Predicting the Outcome of Extracorporeal Shock Wave Lithotripsy for Urinary Calculi. BJU Int. 2005, 95, 1285–1288.
59. Fowler, K. A. B.; Locken, J. A.; Duchesne, J. H.; Williamson, M. R. US for Detecting Renal Calculi With

Nonenhanced CT as a Reference Standard. Radiology 2002, 222, 109–113.
60. Levy, F. L.; Adams-Huet, B.; Pak, C. Y. Ambulatory Evaluation of Nephrolithiasis: An Update of a 1980 Protocol. Am. J. Med. 1995, 98, 50–59.
61. Parks, J. H.; Coe, F. L. A Urinary Calcium-Citrate Index for the Evaluation of Nephrolithiasis. Kidney Int. 1986, 30, 85–90.
62. Odvina, C. V.; Sakhaee, K.; Heller, H. J.; Peterson, R. D.; Poindexter, J. R.; Padalino, P. K.; Pak, C. Y. Biochemical Characterization of Primary Hyperparathyroidism With and Without Kidney Stones. Urol. Res. 2007, 35, 123–128.
63. Labrecque, M.; Dostaler, L. P.; Rousselle, R.; Nguyen, T.; Poirier, S. Efficacy of Nonsteroidal Anti-Inflammatory Drugs in the Treatment of Acute Renal Colic: A Meta-Analysis. Arch. Intern. Med. 1994, 154, 1381–1387.
64. Miller, O. F.; Kane, C. J. Time to Stone Passage for Observed Ureteral Calculi: A Guide for Patient Education. J. Urol. 1999, 162, 688–690.
65. Dellabella, M.; Milanese, G.; Muzzonigro, G. Randomized Trial of the Efficacy of Tamsulosin, Nifedipine, and Phloroglucinol in Medical Expulsive Therapy for Distal Ureteral Calculi. J. Urol. 2005, 174, 167–172.
66. Hollingsworth, J. M.; Rogers, M. A.; Kaufman, S. R.; Bradford, T. J.; Saint, S.; Wei, J. T.; Hollenbeck, B. K. Medical Therapy to Facilitate Urinary Stone Passage: A Meta-Analysis. Lancet 2006, 368, 1171–1179.
67. Borghi, L.; Meschi, T.; Amato, F.; Novarini, A.; Romanelli, A.; Cigala, F. Nifedipine and Methylprednisolone in Facilitating Ureteral Stone Passage: A Randomized, Double-Blind, Placebo-Controlled Study. J. Urol. 1994, 152, 1095–1098.
68. Clayman, R. V.; McDougall, E. M.; Nakada, S. Y. Endourology of the Upper Urinary Tract: Percutaneous Renal and Ureteral Procedures. In Campbell's Urology, 6th ed.; Walsh, P. C., Retik, A. B., Stamey, T. A., Vaughan, E. D., Eds.; W.B. Saunders: Philadelphia, PA, 1992; pp 2789–2821.
69. Lingeman, J. E.; Woods, J. R.; Toth, P. D. Blood Coagulation in Stone Patients: Effect of Extracorporeal Shock Wave Lithotripsy. Urology 1994, 43, 559–566.
70. Samplaski, M. K.; Irwin, B. H.; Desai, M. Less-Invasive Ways to Remove Stones From the Kidneys and Ureters. Cleve. Clin. J. Med. 2009, 76, 592–598.
71. Grasso, M.; Ficazzola, M. Retrograde Ureteropyeloscopy for Lower Pole Caliceal Calculi. J. Urol. 1999, 162, 1904–1908.
72. Preminger, G. M.; Tiselius, H. G.; Assimos, D. G.; Alken, P.; Buck, C.; Gallucci, M.; Knoll, T.; Lingeman, J. E.; Nakada, S. Y.; Pearle, M. S.; Sarica, K.; Türk, C.; Wolf, J. S., Jr. 2007 Guideline for the Management of Ureteral Calculi. J. Urol. 2007, 178, 2418–2434.
73. Park, H.; Park, M.; Park, T. Two-Year Experience With Ureteroscopic Manipulation of Ureteral Stones. J. Endourol. 1998, 12, 501–504.
74. Segura, J. W.; Preminger, G. M.; Assimos, D. G.; Dretler, S. P.; Kahn, R. I.; Lingeman, J. E.; Macaluso, J. N., Jr. Ureteral Stones Clinical Guidelines Panel Summary Report on the Management of Ureteral Calculi. J. Urol. 1997, 158, 1915–1921.
75. Albala, D. M.; Assimos, D. G.; Clayman, R. V.; Denstedt, J. D.; Grasso, M.; Gutierrez-Aceves, J.; Kahn, R. I.; Leveillee, R. J.; Lingeman, J. E.; Macaluso, J. N., Jr.; Munch, L. C.; Nakada, S. Y.; Newman, R. C.; Pearle, M. S.; Preminger, G. M.; Teichman, J.; Woods, J. R. Lower Pole I: A Prospective Randomized Trial of Extracorporeal Shock Wave Lithotripsy and Percutaneous Nephrostolithotomy for Lower Pole Nephrolithiasis—Initial Results. J. Urol. 2001, 166, 2072–2080.
76. Netto, N. R., Jr.; Claro, J. F.; Lemos, G. C.; Cortado, P. L. Renal Calculi in Lower Pole Calices: What Is the Best Method of Treatment? J. Urol. 1991, 146, 721–723.
77. Kuo, R. L.; Aslan, P.; Fitzgerald, K. B.; Preminger, G. M. Use of Ureteroscopy and Holmium:YAG Laser in Patients With Bleeding Diatheses. Urology 1998, 52, 609–613.
78. Borghi, L.; Meschi, T.; Maggiore, U.; Prati, B. Dietary Therapy in Idiopathic Nephrolithiasis. Nutr. Rev. 2006, 64, 301–312.
79. Fink, H. A.; Wilt, T. J.; Eidman, K. E.; Garimella, P. S.; MacDonald, R.; Rutks, I. R.; Brasure, M.; Kane, R. L.; Ouellette, J.; Monga, M. Medical Management to Prevent Recurrent Nephrolithiasis in Adults: A Systematic Review for an American College of Physicians Clinical Guideline. Ann. Intern. Med. 2013, 158, 535–543.
80. Curhan, G. C.; Willett, W. C.; Rimm, E. B.; Stampfer, M. J. A Prospective Study of Dietary Calcium and Other Nutrients and the Risk of Symptomatic Kidney Stones. N. Engl. J. Med. 1993, 328, 833–838.
81. Curhan, G. C.; Willett, W. C.; Speizer, F. E.; Spiegelman, D.; Stampfer, M. J. Comparison of Dietary Calcium With Supplemental Calcium and Other Nutrients as Factors Affecting the Risk for Kidney Stones in Women. Ann. Intern. Med. 1997, 126, 497–504.
82. Nouvenne, A.; Meschi, T.; Prati, B.; Guerra, A.; Allegri, F.; Vezzoli, G.; Soldati, L.; Gambaro, G.; Maggiore, U.; Borghi, L. Effects of a Low-Salt Diet on Idiopathic Hypercalciuria in Calcium-Oxalate Stone Formers: A 3-mo Randomized Controlled Trial. Am. J. Clin. Nutr. 2010, 91, 565–570.
83. Laerum, E.; Larsen, S. Thiazide Prophylaxis of Urolithiasis: A Double-Blind Study in General Practice. Acta Med. Scand. 1984, 215, 383–389.
84. Barcelo, P.; Wuhl, O.; Servitge, E.; Rousaud, A.; Pak, C. Y. Randomized Double-Blind Study of Potassium Citrate in Idiopathic Hypocitraturic Calcium Nephrolithiasis. J. Urol. 1993, 150, 1761–1764.
85. Ettinger, B.; Tang, A.; Citron, J. T.; Livermore, B.; Williams, T. Randomized Trial of Allopurinol in the Prevention of Calcium Oxalate Calculi. N. Engl. J. Med. 1986, 315, 1386–1389.
86. Streem, S. B.; Hall, P. Effect of Captopril on Urinary Cystine Excretion in Homozygous Cystinuria. J. Urol. 1989, 142, 1522–1524.
87. Perazella, M. A.; Buller, G. K. Successful Treatment of Cystinuria With Captopril. Am. J. Kidney Dis. 1993, 21, 504–507.
88. Eckel, R. H.; Grundy, S. M.; Zimmet, P. Z. The Metabolic Syndrome. Lancet 2005, 365 (9468), 1415–1428.
89. Wong, Y.; Cook, P.; Roderick, P.; Somani, B. K. Metabolic Syndrome and Kidney Stone Disease: A Systematic Review of Literature. J. Endourol. 2016, 30 (3), 246–253.
90. 90. Sakhaee, K. Recent Advances in the Pathophysiology of Nephrolithiasis. Kidney Int. 2009, 75 (6), 585–595.
91. Sakhaee, K.; Maalouf, N. M.; Sinnott, B. Clinical Review: Kidney Stones 2012: Pathogenesis, Diagnosis, and Management. J. Clin. Endocrinol. Metab. 2012, 97 (6), 1847–1860.
92. Daudon, M.; Traxer, O.; Conort, P.; Lacour, B.; Jungers, P. Type 2 Diabetes Increases the Risk for Uric Acid Stones. J. Am. Soc. Nephrol. 2006, 17, 2026–2033.
93. Maalouf, N. M.; Cameron, M. A.; Moe, O. W.; Adams-Huet, B.; Sakhaee, K. Low Urine pH: A Novel Feature of the Metabolic Syndrome. Clin. J. Am. Soc. Nephrol. 2007, 2, 883–888.
94. Obligado, S. H.; Goldfarb, D. S. The Association of Nephrolithiasis With Hypertension and Obesity: A Review. Am. J. Hypertens. 2008, 21, 257–264.

95. Taylor, E. N.; Curhan, G. C. Differences in 24-Hour Urine Composition Between Black and White Women. J. Am. Soc. Nephrol. 2007, 18, 654–659.
96. West, B.; Luke, A.; Durazo-Arvizu, R. A.; Cao, G.; Shoham, D.; Kramer, H. Metabolic Syndrome and Self-Reported History of Kidney Stones: The National Health and Nutrition Examination Survey (NHANES III) 1988–1994. Am. J. Kidney Dis. 2008, 51 (5), 741–747.
97. Taylor, E. N.; Stampfer, M. J.; Curhan, G. C. Diabetes Mellitus and the Risk of Nephrolithiasis. Kidney Int. 2005, 68, 1230–1235.
98. Madore, F.; Stampfer, M. J.; Rimm, E. B.; Curhan, G. C. Nephrolithiasis and Risk of Hypertension. Am. J. Hypertens. 1998, 11 (1), 46–53.
99. Abdulla, A.; Das, S.; Venugopalan, A. V.; Rajeevan, A. T.; Manikandan, M. The Correlation Between Metabolic Syndrome and Calcium Urolithiasis. Int. Surg. J. 2023, 10 (9), 1477–1483.
100. Rule, A. D.; Bergstralh, E. J.; Melton, L. J., III; Li, X.; Weaver, A. L.; Lieske, J. C. Kidney Stones and the Risk for Chronic Kidney Disease. Clin. J. Am. Soc. Nephrol. 2009, 4, 804–811.